RELEASE OF MEDIATORS TYPICAL OF SYSTEMIC INFLAMMATORY RESPONSE SYNDROME IN THE COURSE OF A DELAYED HEMOLYTIC TRANSFUSION REACTION

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Published in vitro studies suggest that mediators of systemic inflammatory response syndrome (SIRS) are generated in the course of hemolytic transfusion reactions. Evidence for the in vivo significance of these findings is given by the present clinical and laboratory analysis of a severe delayed hemolytic transfusion reaction (DHTR). A 67-year-old patient (0 Rh-negative) with a negative pretransfusion antibody screen received a massive transfusion because of arterial bleeding (day 1). It was unavoidable to transfuse 0 Rh-positive erythrocyte concentrates because of limited supplies. At day 10 the patient developed a DHTR with symptoms of a septic-toxic syndrome and signs of hemolysis; he received an exchange transfusion. Serological markers, as well as pro- and antiinflammatory mediators were monitored at the onset of the DHTR and during the exchange transfusion. At day 10 the direct antiglobulin test was positive; anti-D was present, most likely as the result of an anamnestic immune response. IL-1 was not detectable; all other mediators monitored were elevated: IL-1ra, TNF, IL-6, IL-8, IL-10, neopterin, elastase, C3a-desArg, C-reactive protein and fibrinogen. Most of the parameters declined during the exchange transfusion, followed by an improvement of the clinical presentation.

Conclusions: Mediators of SIRS were released in the course of a delayed hemolytic transfusion reaction caused by anti-D. Severe clinical symptoms could successfully be treated by exchange transfusion.
We studied biochemical and rheological parameters of stored packed red cells from placental blood. A total of 59 whole blood collections in CPDA-1 were taken immediately after cord clamping and processed within 6 hours. Plasma anduffy coat were removed and packed red cells were stored as unchanged in PAGGS-M. 

Mean corpuscular volumes (MCV) were determined automatically (Celldye 1600, Abbott). Significant cell swelling was observed within 28 days of storage in CPDA-1 and SAG-M, whereas MCV remained unchanged in PAGGS-M.

Viscosities were determined by capillary viscosimetry at adjusted hematocrits of 0.40 U. Viscosities in narrow tubes (270 and 87 μm) increased significantly in CPDA-1 preparations, but remained unchanged in SAG-M and PAGGS-M preparations over the entire storage period.

...2,3DPG was determined enzymatically. Concentrations (mean values at day 28 in % of initial) decreased significantly in CPDA-1 (8.4%), SAG-M (15.4%) and PAGGS-M (19.5%) within 28 days.

Results: Infection rate (pneumonia, wound infection, sepsicaemia, positive pretransfusion antibody screen) increased in patients with transfusions because of arterial bleeding (day 1). It was unavoidable to transfuse 0 Rh-positive erythrocyte concentrates because of limited supplies. At day 10 the patient developed a DHTR with symptoms of a septic/toxic syndrome and signs of hemolysis; he received an exchange transfusion. Serological markers, as well as pro- and antinflammatory mediators were monitored at the onset of the DHTR and during the exchange transfusion. The course of a proinflammatory response syndrome (SIRS) were generated in the course of hemolytic transfusion reactions. Evidence for the in vivo significance of these findings is given by the present clinical and laboratory analysis of a severe delayed hemolytic transfusion reaction (DHTR). A 67-year-old patient (0 Rh-negative) with a negative pretransfusion antibody screen received a massive transfusion because of arterial bleeding (day 1). It was unavoidable to transfuse 0 Rh-positive erythrocyte concentrates because of limited supplies. At day 10 the patient developed a DHTR with symptoms of a septic/toxic syndrome and signs of hemolysis; he received an exchange transfusion. Serological markers, as well as pro- and antinflammatory mediators were monitored at the onset of the DHTR and during the exchange transfusion. At day 10 the direct antiglobulin test was positive; anti-D was present, most likely as the result of an anamnestic immune response. IL-1 was not detectable; all other mediators monitored were elevated: IL-1α, TNF-α, IL-6, IFN-γ, IL-10, neopterin, elastase, C3a-desArg, C-reactive protein and fibrinogen. Most of the parameters declined during the exchange transfusion, followed by an improvement of the clinical presentation.

Conclusions: Mediators of SIRS were released in the course of a delayed hemolytic transfusion reaction caused by anti-D. Severe clinical symptoms could successfully be treated by exchange transfusion.